INCIPIENT SYMMETRICAL PERIPHERAL GANGRENE COMPLICATING PAROXYSMAL TACHYCARDIA

BY

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Bilaterally symmetrical incipient gangrene of all the bodily extremities is sufficiently rare to merit the report of the following case.

Case Report

Mrs. H., a housewife, aged 47 years, was admitted to hospital in January 1947. She had been married 27 years, and had two healthy adult sons. Three days previously she was suddenly taken ill with diarrhœa and sickness, accompanied by faintness and a "fluttering feeling around the heart." Her doctor recommended her admission to hospital because her pulse was extremely rapid, and because her general condition deteriorated swiftly. Her history revealed two similar attacks, the last six weeks previously. This began with temporary loss of consciousness and lasted about two days, and on the second day she noticed that her hands and feet were very cold. However, she soon made an uneventful recovery. For the last year she had been conscious of a fluttering in the chest on exertion, which had lately been lasting longer and had been more easily provoked.

On admission, she was obviously a very ill woman. The hands, feet, nose and ears were very cold, extremely cyanosed, painful, but anæsthetic to light touch. The skin of the extremities did not blanch on pressure. The radial, ulnar, and dorsalis pedis pulses were imperceptible. The brachial and popliteal pulses were palpable. The systolic blood pressure was 65 mm. in the arms, and 85 mm. in the legs; the diastolic was unobtainable. The pulse rate was extremely rapid. Clinically, the heart was enlarged to right and left, but the limits were difficult to define. An apical thrill and murmur were noted, but they could not be timed owing to the tachycardia. The neck veins were markedly distended and

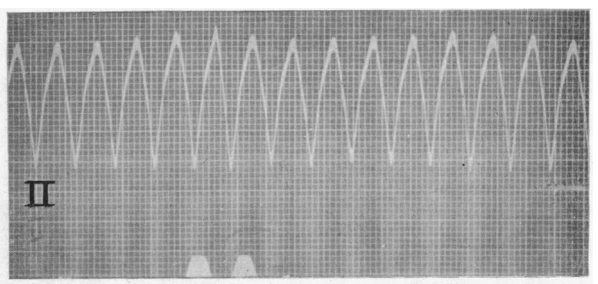

Fig. 1.—Ventricular tachycardia, rate 230 a minute.

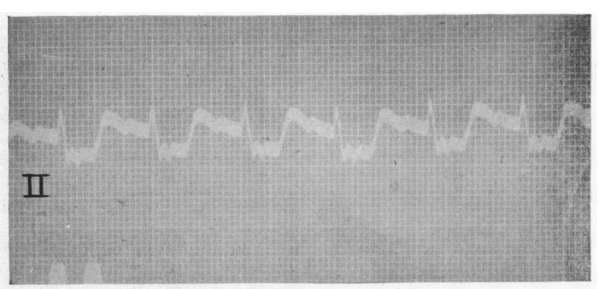

Fig. 2.—Normal rhythm, rate 97, P–R interval 0·16 sec., S–T segment depressed.

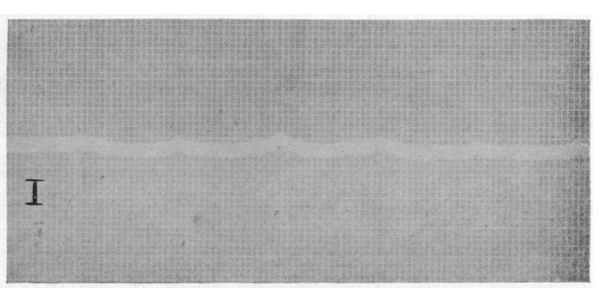

Fig. 3.—Ventricular asystole, ventricular rate 90.

quivering, being engorged up to the ears, whilst the arm veins were so empty of blood that they could not be seen at all. The liver was just palpable; ascites and pitting œdema were absent, but there were a few moist sounds at the lung bases. Throughout all this time the patient remained co-operative, calm, alert, and in full possession of her mental faculties.

A portable chest X-ray provided no useful information. An electrocardiogram showed a regular paroxysmal ventricular tachycardia at a rate of 230 a minute (Fig. 1).

Thus the clinical picture was that of extreme circulatory failure, of which the immediate precipitating cause was the tachycardia. It was imperative to slow the heart rate and to raise the blood pressure as soon as possible, and quinidine was given intravenously for this purpose. After a test dose of 3 grains had excluded idiosyncrasy to the drug, a solution containing one gram of quinidine sulphate in 100 ml. of glucose saline was administered by slow intravenous drip. Half an hour later, when the patient had received approximately 60 ml. of the solution (0·6 g. quinidine), she suddenly became distressed and lost consciousness. The drip was discontinued and a further electrocardiogram was taken (Fig. 2). This was thought to show regular rhythm at a rate of 97 a minute; the QRS complexes were less widened. A period of ventricular asystole then ensued, with an auricular rate of 90 a minute (Fig. 3). This was followed by cardiac standstill and death four hours after her admission.

Post-mortem examination. The cardiovascular system alone showed relevant pathological changes. There was extensive syphilitic aortitis, with aneurysmal dilatation of the ascending part and arch of the aorta. The coronary arteries were

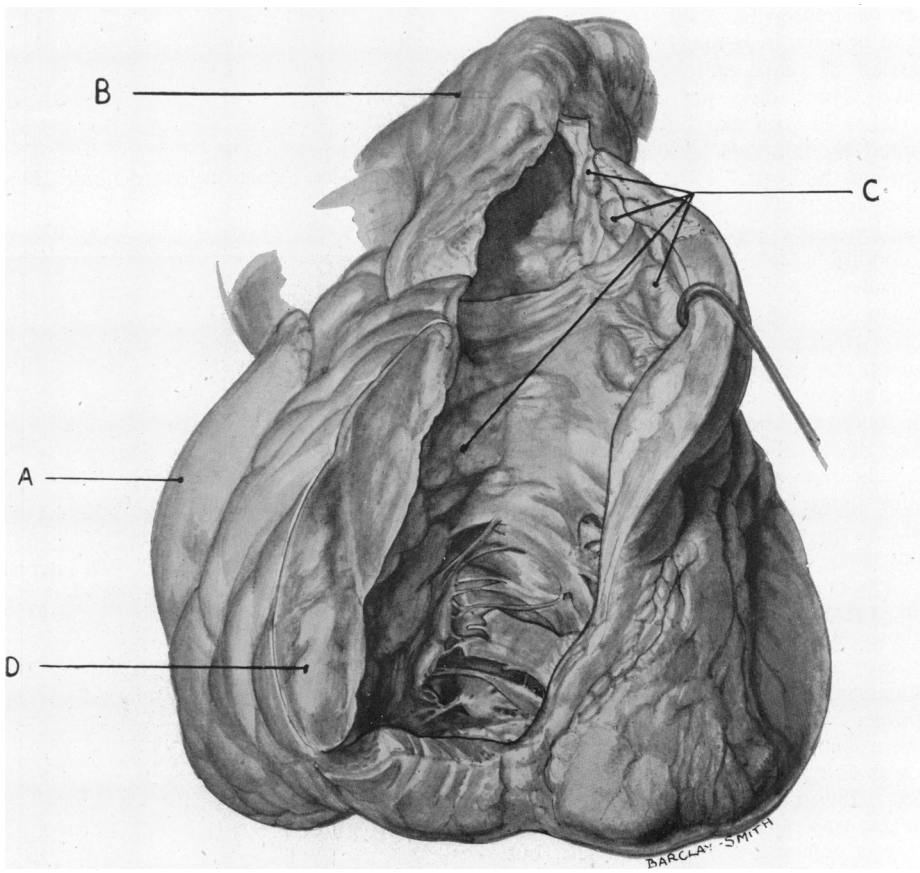

FIG. 4.—Drawing of heart with right ventricular cavity open. (A) Right auricle, (B) Pulmonary artery, (C) Granulomatous masses in right ventricle and pulmonary artery, and (D) Gummatous infiltration of the wall of the right ventricle.

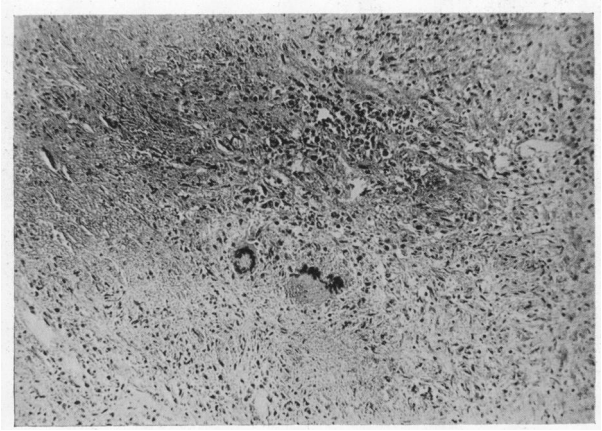

FIG. 5.—Gumma of myocardium. Magnification ×125.

healthy and showed no sign of narrowing of their orifices. The left auricle and ventricle appeared normal, but the muscle of the right ventricle and also the interventricular septum showed diffuse fibrotic infiltration. There were, in addition, numerous large, discrete, granulomatous lesions distributed over the internal surface of the right ventricle; one particular mass lying under the tricuspid valve measured three-quarters of an inch in diameter. The pulmonary artery was similarly affected (Fig. 4). The arteries to the upper and lower extremities showed no abnormality throughout their lengths. Section and microscopic examination revealed that both the diffuse infiltration and the granulomatous masses were syphilitic in nature (Fig. 5 and 6).

DISCUSSION

The case presents several points of interest. Gumma of the heart is comparatively rare. In fourteen years of extensive study of syphilitic cardiovascular disease, Maynard (1943) stated that this lesion had been encountered only once at necropsy. When present, gummata usually occur in the left ventricular myocardium and particularly in the basal portion of the septum (Sohval, 1935). Sohval also states that diffuse gummatous myocarditis is extremely rare by itself, being usually associated with gumma of the heart, as it was in this case. Syphilis of the pulmonary artery is also rare, though in a review of cases showing aneurysm of the pulmonary artery, Boyd (1941) stated that syphilis was the ætiological factor in thirty-three cases (31 per cent).

A paroxysm of ventricular tachycardia usually lasts no more than an hour (Cooke and White, 1943), but longer attacks, lasting 1 to 3 weeks have been recorded (Dubbs and Parmet, 1942; Beers and de la Chapelle, 1947). In the present case, the paroxysm lasted just over three days.

Symmetrical peripheral gangrene is mentioned by Fishberg (1944) as occurring in occlusion of the mitral valve by a ball thrombus, and in massive cardiac infarction. I can find no other references concerning its occurrence in cardiac infarction, but Abramson (1924) recorded it in a review of ball thrombi of the heart. He stated that severe disturbances of the general circulation were the rule, and that gangrene of the extremities sometimes occurred, which he thought was usually due to peripheral arterial emboli. Tight or button-hole stenosis of the mitral valve frequently gives rise to intense cyanosis, and coldness of the extremities, but not to gangrene.

Fishberg (1938) noted the co-existence of "collapsed" veins in the extremities with engorged jugular veins, in extreme failure of the right heart. This he attributed to compensatory vaso-constriction in the extremities evoked reflexly by extreme diminution in cardiac output. In this connection he observed symmetrical peripheral incipient gangrene on two occasions.

Bruce Perry and Davie (1939) reported bilateral symmetrical peripheral incipient gangrene in the lower limbs in a case of hypertensive heart disease, terminating in congestive failure, and agreed with Fishberg that excessive peripheral vasoconstriction was responsible. Chatterjee (1940) noted peripheral symmetrical incipient gangrene during the course of

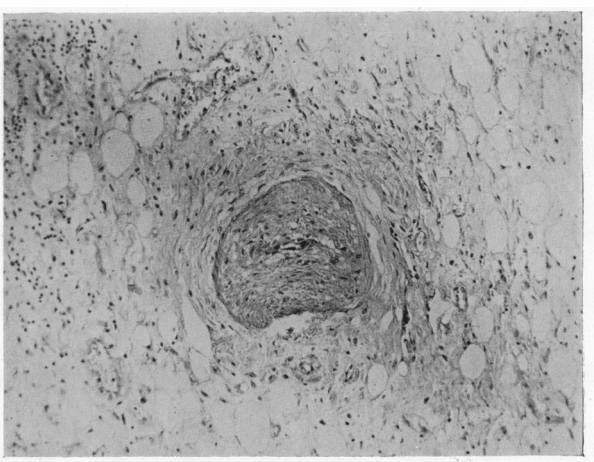

FIG. 6.—Obliterating endarteritis of myocardial vessel. Magnification ×125.

lobar pneumonia, and concluded that it was due to acute circulatory failure, probably reflex in origin.

In the present case, neither arterial emboli, nor luetic arteritis played any local part in the genesis of the gangrene. It seems certain, therefore, that reflex peripheral vasospasm, as indicated by Fishberg, was responsible, and that this was evoked by a critically low cardiac output, due to persistent ventricular tachycardia.

Summary and Conclusions

A case of incipient symmetrical peripheral gangrene is described, associated with extreme heart failure due to persistent ventricular tachycardia. Necropsy revealed the presence of discrete gummata of the heart and pulmonary artery, and diffuse gummatous infiltration of the right ventricle and interventricular septum. Similar cases are briefly reviewed, and it is concluded that the incipient gangrene was due to compensatory peripheral vasoconstriction.

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